Ventricular septal defects in cattle: A retrospective study of 25 cases

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Abstract — Clinical and complementary examination, pathological findings, and outcome were reviewed for 25 heifers diagnosed with ventricular septal defect (VSD). Age at presentation ranged from 36 h to 21 mo. The most common reasons for consultation were chronic respiratory problems (11 cases; 44%) or assessment of heart murmur (5 cases; 20%). A pansystolic murmur on the right thoracic side was audible in 20 cases (80%). Pneumonia that interfered with adequate cardiac auscultation was found in 15 calves (60%). Echocardiography was useful in establishing the final diagnosis of VSD (sensitivity of 94%). Prognosis was poor: only 10 calves were discharged and no heifer out of the 6 cases for which follow-up information was available had a productive life in the herd. Inadequate reproductive performance was owners' most common complaint (4 of 6 heifers).

Résumé — Communications interventriculaires chez le veau : une étude rétrospective de 25 cas. Les données cliniques, les examens complémentaires, et le devenir de 25 génisses atteintes de communication inter-ventriculaire (CIV) ont été étudiées. La raison principale de référence était un problème respiratoire chronique (11 cas; 44 %) ou l'évaluation d'un souffle cardiaque (5 cas; 20 %). Une infection pulmonaire a été identifiée chez 15 animaux (60 %), ce qui pouvait interférer avec l'auscultation. Un souffle pansystolique d'intensité supérieure à droite a été audible chez 20 animaux (80 %). L'échocardiographie s'est avérée un moyen fiable de confirmer le diagnostic des CIV (sensibilité de 94 %). Le pronostic à long terme de ces animaux a été jugé pauvre. Seuls 10 animaux ont obtenu leur congé de l'hôpital. Aucun animal, parmi les 6 pour lesquels un suivi était disponible, n'a eu de vie productive dans l'élevage. Les problèmes de reproduction constituaient la plainte majeure des propriétaires (4 des 6 génisses). Malheureusement, aucune donnée clinique, ou échographique n'est apparue utile pour établir un pronostic selon cette étude.

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Introduction

Congenital cardiac defects are relatively rare in cattle, being observed in only 0.17% of 50 742 bovine hearts inspected in a slaughterhouse study (1). In a retrospective study of 2293 calves with congenital anomalies, cardiac malformations occurred in 2.7% of the cases (2). Ventricular septal defect (VSD) is the most common cardiovascular abnormality in bovine neonates (1,3–6). It was found in 11 of 36 and 7 of 10 calves diagnosed with heart defects in 2 separate studies (2,6). Ventricular septal defect in cattle has also been identified in combination with multiple cardiac defects, such as dextroposition of the aorta (1,6–11), patent ductus arteriosus (6,7,12), persistent foramen ovale (6,9,13), transposition of the great vessels (6,7), persistent truncus arteriosus (10,11,13), and anomaly of the pulmonary (7,11) or tricuspid valve (6). On the right side of the heart, the anatomic position of the defect is described by its relative position to the crista supraventricularis muscular ridge. On the left, the defect is most frequently located in the membranous part of the interventricular septum, adjacent to the aortic valve (14,15), and it causes free shunting of blood between the ventricles. Blood from the left ventricle is shunted towards the right ventricle and pulmonary arteries. The excessive volume-flow overloads the pulmonary vascular tree and can lead to pulmonary hypertension (16–18). Since shunting occurs from left to right, the left heart demonstrates morphological changes typical of volume-flow overload (left atrial and left ventricle enlargement). When the pulmonary pressure exceeds the systemic pressure, reversal of the shunt may occur. That phenomenon is called the Eisenmenger's complex. Calves with a VSD usually had clinical signs of heart failure (6–8,10–13); in some situations, the condition may occur without any clinical signs other than a cardiac murmur (9,19). Ventricular septal defect, as with other congenital heart defects in humans (20) and small animals (17), can damage the endocardium by blood flow turbulences and predispose to bacterial endocarditis. This association has not been clearly demonstrated in cattle. Since there are limited data on the clinical signs, concomitant findings, and outcome in cattle with a VSD, a study of the clinical picture of VSD in cattle appears to be appropriate.

The objective of the present study was to describe the findings and the outcome in cattle that were referred to a veterinary teaching hospital with a VSD. The history, physical examination findings, results of ancillary procedures, and outcome of the cases diagnosed as having a VSD at the Centre Hospitalier Universitaire Vétérinaire (CHUV) de l'Université de Montréal are reported here.

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Materials and methods

The medical records of all cattle presented at the CHUV between 1987 and 2003 were reviewed and selected on the basis of a final diagnosis of VSD. Cattle with tetralogy or pentalogy of Fallot were excluded, since they were considered to be different clinical (16,17) and embryological entities (21). Cattle without ultrasonographic or postmortem confirmation of VSD were also excluded. Ultrasonographic diagnosis was based on visualization of the defect in the ventricular septum when the heart was imaged in the long axis using 2D and color flow (CF) Doppler (14,19).

For each case, the history, breed, sex, age, and suspected diagnosis by the referring veterinarian were extracted from the medical records. Results of the clinical examination and cardiorespiratory function were also reviewed. Results of the arterial blood gases obtained by auricular artery puncture and the complete blood (cell) count (CBC) performed on the day of admittance were studied when available. Echocardiographic data concerning the size of the defect and its location in the interventricular septum, the presence of other cardiac anomalies, the electrocardiographic findings, and the pulmonary radiographs were also reviewed. The mean size of the defect, as measured by ultrasonography, was compared by a chi-squared test in heifers that were discharged and the group that died or were euthanazied. The difference was considered significant if $P < 0.05$.

Results of pathologic findings, with emphasis on the size and the location of the VSD (membranous or muscular), other concomitant congenital anomalies, or presence of an infectious process, were examined when available. The owners of calves that were discharged were contacted by telephone to obtain follow-up information. Details about outcome of the animal, reason for culling or death, level of production, and the presence of other congenital abnormalities in the herd were also investigated.

Results

Of the 35 medical files initially identified, 10 were excluded since they were lacking ultrasonographic or pathologic confirmation of VSD. Only 2 of the remaining animals were reassessed 8 and 17 mo after diagnosis. All animals were female, Holstein (n = 22) and Ayrshire (n = 3) were the only 2 breeds represented. Age ranged from 36 h to 21 mo (median = 2.0 mo). The clinical picture of each heifer is described in Table 1.

The most common reason for presentation was respiratory problems (11/25; 44% cases) and assessment of a heart murmur (5/25; 20% cases). Other uncommon presenting complaints included undetermined cardiorespiratory dysfunction (n = 4), diarrhea (n = 4), anorexia (n = 4), dysphagia (n = 3), bloating (n = 2), omphalophlebitis (n = 1), vaginal bleeding (n = 1), and head tilt (n = 1).

Clinical findings in affected heifers revealed poor growth in 3 cases. Tachycardia (n = 17), and tachypnea and hyperthermia (n = 16) were common findings (Table 1). A pansystolic murmur of loudest intensity on the right thoracic side with a palpable thrill was the most frequent physical observation (n = 20). In the 5 remaining cases, no murmur was detected. Clinical signs of cardiorespiratory failure, including respiratory distress (n = 4); depression (n = 4); increased capillary refill time (n = 4); peripheral edema, distended jugular veins, or elevated jugular pulse (n = 4); cyanosis (n = 3); and arrhythmia (n = 1), were not as frequently encountered. Pulmonary auscultation was often abnormal with pulmonary wheezes or crackles (n = 8). A cough was noted in only 3 cases and it was associated with pneumonia.

Hematological findings were available for 23 cases; abnormal results have been noted in Table 2. Packed cell volume (PCV) was lower than normal in 5 cases (range 0.22 to 0.26 L/L; mean = 0.25 L/L). The most common abnormality was neutrophilia in 13 cases (range: 4.8 to 42.9×10^9 cells/L; mean = 14.7×10^9 cells/L reference range, 0.6 to 4×10^9 cells/L), increased fibrinogen concentration in 7 calves (range: 6 to 8 g/L; mean = 6.9 g/L reference range, 3 to 5 g/L) and monocytosis in 7 cases (range: 1.2 to 3.5×10^9 cells/L; mean = 1.8×10^9 cells/L reference range, 0.0 to 0.8×10^9 cells/L). All cases with hyperfibrinogenemia (n = 7) and 5 of the 7 cases with monocytosis had a pneumonia. Two of the 5 anemic cases had pneumonia. Biochemical profiles were unremarkable. Arterial blood gases, available in 11 cases, revealed mild to severe hypoxia (range: 78.1 to 25.6 mmHg; mean = 53.5 mmHg reference range 90 to 110 mmHg) in 8 cases, and hypercapnia (range: 45.9 to 52.4 mmHg; mean = 49.1 mmHg reference range, 40 to 45 mmHg) in 5 cases. These blood gas abnormalities during hospitalization were always associated with a poor outcome (natural death or euthanasia).

Thoracic radiographs were taken in 17 animals (Table 2). An alveolar pattern located in the ventral pulmonary field was identified in 15 cases (88%); a bronchial and an interstitial pattern were found in 5 and 6 calves, respectively. Cavitary lesions, interpreted as possible pulmonary abscesses, were found in 5 cases. Evidence of cardiomegaly was identified in only 2 cases, with important enlargement of the heart compared with that of contemporary heifers. Other radiographic findings were pleural effusion in 1 heifer, multiple rib fractures in 1 neonate, and a hypoplastic trachea in 1 heifer. Carotid angiography revealed an aneurysm located in the right carotid artery in heifer #11. Radiographs of the tympanic bulla were performed in 1 calf with head tilt and they indicated osteolysis and osteoproliferation of the right tympanic bulla, compatible with otitis interna/media.

To investigate the cause of detected heart murmurs, echocardiography was performed in 17 heifers, and the presumptive clinical diagnosis of VSD was confirmed in 16. The heifer misdiagnosed by echocardiography had a large mass on the left atrioventricular valve, interpreted as vegetative bacterial endocarditis, but no VSD was identified. However, postmortem examination revealed a 2 cm wide VSD and marked nodular fibrosis of unknown origin in the mitral valve.

The location of the defect in the septum was inconsistently mentioned in the ultrasonographic reports. The size of the VSD was estimated during echocardiography (n = 13). In 7 of 13 cases (53.8%) defects between 2 and 4 cm in diameter were identified. Three VSDs were

Table 1. Reasons for referral and clinical findings in 25 heifers with a ventricular septal defect (VSD)

Case number	Age (months)	Heart rate (beats per min)	Respiratory rate (breaths per min)	Rectal temperature (°C)	Heart murmur	Other clinical findings
1	21	80	44	38.8	Yes	—
2	0.03 (1 d)	160	140	39.5	Yes	Respiratory distress, ICRT, depression
3	2	144	60	39.3	Yes	ICRT
4	1	80	30	39.3	Yes	Poor growth, cardiac arrhythmia
5	3.5	140	100	41.0	Yes	ICRT
6	0.5	111	40	40.3	Yes	Cough, ICRT
7	8.5	152	54	40.8	No	Depressed
8	0.5	132	92	40.1	Yes	Depressed, cyanosis, ARN
9	0.5	54	32	<37	No	Shock, cyanosis, respiratory distress
10	4	112	48	39.8	Yes	—
11	0.06 (2 d)	110	88	40.3	Yes	Jugular pulse, cleft palate, ARN, diarrhea, mass in the brisket area
12	0.06 (2 d)	150	114	39.0	Yes	Depressed, cough, cyanosis, ARN, respiratory distress
13	2.5	120	36	39.6	Yes	Poor growth
14	3	76	30	38.6	Yes	—
15	2.25	200	69	40.4	No	ARN, jugular distension, brisket edema, abdominal distension
16	3.25	108	60	39.3	No	Poor growth, ARN, cough
17	6	100	36	39.0	Yes	Free gas bloat
18	1.5	120	30	39.8	Yes	Head tilt, ARN
19	2	152	60	40.1	Yes	ARN
20	1	160	135	39.7	Yes	—
21	0.05 (1.5 d)	140	120	39.8	Yes	Respiratory distress
22	0.06 (2 d)	160	52	39.6	No	—
23	0.12 (4 d)	200	160	42.0	Yes	ARN, respiratory distress
24	3	120	44	39.6	Yes	Jugular pulse, free gas bloat
25	2	136	112	40.3	Yes	Jugular distension

ICRT — Increased capillary refill time; ARN — Abnormal respiratory noises

smaller than 1 cm, and 2 others ranged between 1 and 2 cm. The last calf (#8, Table 2) for which the VSD size was estimated had total absence of the interventricular septum, confirmed at necropsy (12 by 2 cm, Table 3). There was no significant difference between the mean ultrasonographic size of the defect in discharged heifers (mean = 1.59 cm; $n = 6$) and necropsied heifers (mean = 1.92 cm; $n = 6$) ($P = 0.67$). Eight calves for which necropsy findings were available had had a previous echocardiographic examination. The ultrasonographic and necropsic findings are depicted in Tables 2 and 3. The most striking differences were important variations in the size of the defect between the ultrasonographic and necropsy measurements (more than 100% of error in 3 of 6 heifers). Aortic regurgitation was not mentioned in any ultrasonographic report.

In 2 cases, Eisenmenger's complex was diagnosed with reversal of the shunt (#12 and #16). Two cases were assessed 2 times by ultrasonography. Heifer #19 had been examined 17 mo earlier and diagnosed with an uncomplicated VSD (Table 3). According to the owner, she was smaller than expected for her age and was pregnant (60 d) when reexamined. She had been referred to the CHUV because she was recumbent. Examination revealed no murmur, but hypoxia (PaO_2 = 60.4 mmHg), mild polycythemia (PCV = 0.47 L/L) and an abscess at the thoracolumbar vertebral junction. Echocardiography revealed a large VSD (4.5 cm in diameter versus 2.6 cm at the 1st examination) with evidence of pulmonary hypertension and a right to left shunt. Heifer #24 was reexamined 8 mo after the initial diagnosis of VSD. Echocardiography revealed an enlarged VSD of 2.2 to 3.5 cm. The increased size of the defect was not associated with any clinical signs. Abdominal ultrasonography performed in heifer #15 revealed free abdominal fluid interpreted as ascites.

Electrocardiograms were available in 5 heifers and no specific abnormalities were detected. Heifer #11 had right atrial and pulmonary trunk catheterization to identify a gradient in oxygen content between the right atrium and the pulmonary trunk, which confirmed the diagnosis of VSD. Other diagnostic tests performed included: transtracheal aspirate ($n = 4$), fecal egg count ($n = 4$), and blood culture ($n = 2$).

Ten heifers were discharged from the CHUV. On the basis of clinical, hematologic, transtracheal aspirate, and radiographic data, bronchopneumonia was suspected in 7 heifers and antimicrobial therapy was prescribed. Eleven of 12 heifers that were presented with clinical signs of cardiorespiratory failure (respiratory distress, peripheral edema, distended jugular vein or elevated jugular pulse, cyanosis, and increased capillary refill time) died or were euthanized. Necropsy was performed on 15 calves. Cardiac anomalies were identified in all cases. They were associated with variable lung abnormalities ($n = 11$). Bronchopneumonia was the most common pulmonary complication ($n = 8$). Noninfectious pulmonary lesions consisted of pulmonary edema ($n = 1$), atelectasia ($n = 1$), and arteriosclerosis ($n = 1$). Abnormalities associated with congestive heart failure were present in 10 heifers with hydropericardium ($n = 6$), hepatic congestion ($n = 5$), ascites ($n = 3$), and pulmonary edema ($n = 1$). Ventricular septal defect was the only cardiac anomaly in 2 heifers. It was associated with other cardiac abnormalities in the remaining 13 heifers; right ventricular hypertrophy ($n = 6$), dextroposition of

Table 2. Abnormal ancillary test results in 25 heifers with a ventricular septal defect (VSD)

Case number	Complete blood cell count	Arterial blood gas	Thoracic radiographs	Echocardiography
1	Normal	NP	NP	NP
2	↗WBC (21.4); ↗Neu (18.2); ↗Mono (1.24), ↘PCV (0.25)	NP	NP	NP
3	↗WBC (16.9); ↗Neu (9.8)	NP	Cardiomegaly, Alv pattern	VSD
4	↗Neu (5.5)	NP	Ventral Alv pattern	VSD (1 cm)
5	↗Neu (6.1)	↘PaO_2 (52.0)	Alv and Int pattern, CL	NP
6	↗WBC (16.1); ↗Neu (9.3), F = 6 g/L	NP	Ventral Alv pattern	VSD (1.9 cm)
7	↗WBC (47.7); ↗Neu (42.9), F = 8 g/L, ↘PCV (0.24)	NP	Ventral Alv pattern, CL, pleural effusion	VSD (2.2 cm), Dilation of the pulmonary trunk
8	F = 6 g/L	↘PaO_2 (31.6)	NP	VSD, total absence of the septum
9	NP	NP	NP	NP
10	Normal	↘PaO_2 (78.1), ↗$PaCO_2$ (45.9)	Bronc pattern	VSD
11	↗WBC (13.8), ↗PCV (0.26)	↗$PaCO_2$ (52.4)	Ventral Alv pattern; cardiomegaly	No VSD seen, mitral mass
12	↗Neu (12.2)	↘PaO_2 (25.6)	Ventral Alv pattern, Bronc pattern	VSD (0.5 cm), Eisenmenger's complex
13	F = 7 g/L	NP	Ventral Alv pattern	VSD (0.25 cm)
14	↗Neu (7.6), ↗Mono (2.19)	NP	Ventral Alv pattern	VSD (2.0 cm), RVH
15	↗Mono (1.3), ↘PCV (0.22)	NP	NP	VSD (3 cm)
16	↗Neu (4.9)	↘PaO_2 (59.1)	Alv and bronc patterns	VSD (2 cm), Eisenmenger's complex
17	↘PCV (0.26), ↗Mono (1.2)	NP	NP	VSD
18	↗Neu (4.8), ↗Mono (1.2), F = 7 g/L	NP	Alv pattern	NP
19	Normal	Normal	Diffuse Int and Alv patterns	VSD (2.6 cm)
20	NP	NP	NP	VSD
21	↗WBC (43.4), ↗Neu (38.3), ↗Mono (3.5)	↘PaO_2 (50.7), ↘$PaCO_2$ (51.7)	Int and Alv patterns, CL, hypoplastic trachea	VSD (2 cm)
22	↗WBC (29.4), ↗Neu (23.5), F = 7 g/L	↘PaO_2 (67.3), ↗$PaCO_2$ (46.5)	Alv, Bronc, Int patterns, CL, ribs fractures	NP
23	Normal	↗PaO_2 (63.4)	NP	NP
24	↗WBC (18.4), ↗Neu (7.5), ↗Mono (1.3), F = 7 g/L	NP	Int and Alv patterns	VSD (2.2 cm), PFO
25	Normal	↗$PaCO_2$ (49.0)	Alv, Bronc, Int patterns, CL	VSD (1.35 cm), tricuspid fibrosis

NP — not performed; WBC — white blood cells ($\times 10^9$ cells/L); Neu — neutrophils ($\times 10^9$ cells/L); Mono — monocytes ($\times 10^9$ cells/L); PCV — packed cell volume (L/L); F — fibrinogen; Alv — alveolar; Bronc — Bronchial; Int — interstitial; CL — cavitary lesions; PaO_2 — arterial pressure of oxygen (mmHg); $PaCO_2$ — arterial pressure of carbon dioxyde (mmHg); RVH — right ventricular hypertrophy; PFO — patent foramen ovale

the aorta (n = 3), patent ductus arteriosus (n = 3), pulmonary trunk dilation (n = 3), right atrial dilation (n = 2), left ventricular dilation, and patent foramen ovale (n = 1). The exact anatomic location of the VSD was not available. In all necropsy reports, the location of the VSD was in the upper part of the membranous septum near the aortic root, except in heifer #8 with a partial absence of the muscular septum. It was conclude that in 14 heifers a membranous defect was present; in 1 heifer, a muscular VSD was diagnosed. Other congenital defects identified included carotid aneurysm and cleft palate in 1 heifer and hydrocephalus in another. The endocardium was damaged in only 2 heifers (heifers #8 and #11, Table 3). In these cases, focal endocardial fibrosis was noted. However, there were no histopathologic signs of endocarditis.

Follow-up information was obtained for 6 out of the 10 heifers discharged. The size of their VSD varied from 0.25 to 2.6 cm (mean = 1.7 cm). One calf died suddenly shortly after discharge. Four out of the 5 other heifers were culled or died before the age of 2 y. In 4 out of the 6 heifers, they were smaller compared with heifers of a similar age. Reason for early culling was failure to become pregnant in 4 of the 6 cases.

Discussion

Ventricular septal defect is a congenital defect for which the precise etiology is still under investigation in humans (20,21), horses, and cattle (14). In humans, chromosomical abnormalities are associated with VSD (Trisomy 13, or an abnormality of the short extremity of chromosome 5 [20]). However, other factors predispose human fetuses to VSD. Exposure to teratogenic factors during the pregnancy (alcohol) is also associated with VSD (20). In cattle, hereditary transmission is suspected in Herefords (10) and Limousins (5). Genetic predisposition is also reported in Jersey cattle (9). In other dairy breeds, no information is currently available on possible genetic implications; although, VSD was reported in twin Holstein heifers (22). The breed proportions found in the present study reflect the population encountered at the CHUV (majority Holsteins, then Ayrshires, and a few other breeds). Pedigree information of affected cattle was unavailable for evaluation.

Various other congenital defects have been described in cattle with VSD. In our review of the literature, dextroposition of the aorta (1,6–11), patent ductus arteriosus (6,7,12), persistence of the foramen ovale (6,9,13), and persistant truncus arteriosus (10,11,13) were identified

Table 3. Outcome and postmortem examination in 25 heifers with a ventricular septal defect (VSD)

Case number	Necropsy findings	Follow up after discharge
1	VSD, RVH (Heart available)	Culled
2	VSD (1 × 1.2 cm), RVH, HP	—
3	VSD (3 cm), cardiomegaly, pulmonary edema, HP, ascites	—
4	NP	FNA
5	VSD (4 cm), pulmonary atelectasia, hepatic congestion	—
6	NP	Culled (poor breeder and poor growth)
7	NP	FNA
8	Large VSD (12 × 2 cm), RAD, ventricular endocardial fibrosis	—
9	VSD (3 cm), RVH, PTD, pneumonia, HP, hepatic congestion, ascites	—
10	NP	FNA
11	VSD (2 cm), RVH, PTD, mitral and aortic nodular fibrosis, right external carotid aneurysm, cleft palate, pneumonia	—
12	VSD (1.2 cm), PDA, RVH, DextA, Eisenmenger's complex, omphalophlebitis, pneumonia	—
13	NP	Culled (Poor breeder)
14	NP	FNA
15	VSD (2.5 cm), DextA, pulmonary arteriosclerosis, HP, liver congestion, ascites	—
16	VSD (6 cm), PTD, RAD, DextA, Eisenmenger's complex, HP	—
17	NP	Died 2 d after discharge
18	VSD (1 cm), otitis media, synovitis, pneumonia	—
19	Euthanized 17 mo after first diagnosis with Eisenmenger's complex and a vertebral abscess	—
20	NP	Culled (Poor breeder)
21	VSD (2 × 3.5 cm), RVH, LVD, PFO, pneumonia, liver congestion	—
22	VSD (1 cm), PDA, pneumonia, atelectasy, ribs fractures, hydrocephalus	—
23	VSD (2 cm), PDA, aspiration pneumonia	—
24	—	Enlarged VSD 8 mo after initial diagnosis (2.2 to 3.5 cm). Pregnant at 24 mo of age (retarded growth)
25	VSD (3.5cm), pneumonia, hemal nodes on the tricuspid valve, cardiomegaly, HP, liver congestion	—

RVH — right ventricular hypertrophy; HP — hydropericardium; PTD — pulmonary trunk dilation; RAD — right atrial dilation; PDA — patent ductus arteriosus; DextA — dextroposition of the aorta; LVD — left ventricular dilation; PFO — patent foramen ovale; NP — not performed; FNA — follow up not available

and are consistent with human studies showing that the same genotype can induce multiple congenital heart defects (23). Other congenital defects, such as carotid aneurysm and cleft palate, have not been reported in association with VSD.

A loud pansystolic heart murmur of maximal intensity on the right thorax is the most characteristic clinical finding in cattle with VSD (14,22), as is found in humans (24) and horses (14,25). However, this condition may be missed by general practitioners. Optimal auscultation conditions at our hospital allowed us to identify a systolic murmur with maximal intensity on the right thoracic wall and a palpable fremitus in 20 of 25 heifers.

The most common problem associated with VSD was pneumonia, which was diagnosed either at necropsy or on clinical examination since in 11 of 25 heifers, the primary complaint was chronic nonresponsive pneumonia. Pulmonary infection can be a complication of heart failure and pulmonary edema. Impairment of mucociliary clearance has been suspected in asymptomatic cattle suffering from VSD (12,14,19). Infection and the large volume of blood shunting across the VSD and into the pulmonary trunk may contribute to development of pulmonary hypertension (18,26): Eisenmengers complex is a rare but fatal complication of VSD (7,10,19), although, it was diagnosed in 2 heifers by ultrasonography and confirmed by necropsy in the present study. A 3rd heifer developed this complication 8 mo after the initial diagnosis of uncomplicated VSD.

Cough can be a clinical sign of heart disease in humans (20) and dogs (16). However, in this study, it was only observed in the calves with pneumonia. Other clinical signs of cardiac or pulmonary failure, such as respiratory distress, peripheral edema, distended jugular vein or elevated jugular pulse, cyanosis, and increased capillary refill time were also encountered. When such clinical signs were present, the prognosis was fatal in 11 of 12 heifers, and, necropsy findings were compatible with advanced heart disease and in accordance with the poor prognosis of cattle showing signs of heart failure reported in the literature (6–9,11–15).

Bacterial endocarditis is a common complication associated with congenital cardiac defects in humans (24) and small animals (27). The endocardium may sustain damage caused by the turbulent blood flow and becomes more susceptible to bacterial colonization, if transient bacteremia occurs (24). This complication of VSD has been reported in a goat (28) and in a Holstein heifer (22). However, although endocarditis was not identified in the present study, it may have been underestimated because of low survival rate and lack of long-term follow-up. The 2 heifers with focal fibrosis of the endocardium may have been in the first steps of endocarditis development associated with the congenital heart defect (24).

Ventricular septal defect causes a left to right shunt, allowing blood to move freely between ventricles. In humans (24), and horses (25,29), the pathophysiologic consequences of VSD are determined by the size of the

defect and the relative resistance in the systemic and pulmonary vasculatures. Assessment of the shunt peak velocity can be used as a prognostic indicator in some species (16,19,24,29,30). Measurement of blood velocity through the defect or other specific measurements were not consistently available from the medical records in the present study. When pulmonary hypertension is developed, the difference of pressure between left and right heart decreases. The velocity across the defect is correlated with the difference of pressure between the 2 ventricles. In horses, a peak velocity through the VSD from a left to right shunt of more than 4 m/s is compatible with adequate racing performance (29). Dogs with a peak velocity greater than 4.5 m/s have a good prognosis for life (30). A prospective study still needs to be performed to correlate the velocity across the defect and the outcome for affected cattle.

Echocardiography appeared to be a reliable diagnostic tool to detect VSD. Many authors considered it to be the technique of choice for diagnosing VSD in horses (14,19,25) and in small animals (16,30). No specific correlation between the size of the defect and the outcome could be determined in this study. Size of less than 2.5 cm for the larger diameter of the defect is associated with a favorable outcome in horses (29). Our study also showed important differences between ultrasonographic measurements of VSD and postmortem findings, which could complicate the interpretation of the size of the defect as predictor of the outcome (Table 3). Since VSDs are rarely circular, measurement of the defect was of limited value without knowing the exact plane of measurement (29). The use of a standardized ultrasonographic protocol in a prospective study would help to determine the relationship between the size of the defect and the prognosis.

The location of the defect in the septum could only be assessed by using necropsy reports. In the 15 necropsied cattle, only 1 defect was muscular. The others were classified as membranous defects (high in the septum, near the aortic root). This finding is in accordance with other reports in cattle (6,7,10,11,18), in horses (29), and small animals (30). The membranous septum is the site of the embryonic interventricular foramen (20). This type of defect can result from a deficient growth of 1 of the 3 elements that normally close this foramen (atrioventricular endocardial cushion, left bulbar ridge, and interventricular septum) (20). Aortic regurgitation is a complication of VSD in dogs (31) and horses (29), due to the anatomical position of the membranous defect. Occasionally, some degree of prolapse of one cusp of the aortic valve into the VSD can cause significant regurgitation and affect the prognosis (29,31). This event is associated with a diastolic murmur following the systolic murmur of the VSD and can be imaged easily by ultrasonography (16,29–31). It was not identified in any animals in this study. However, it is not known if this condition was investigated in each case.

As previously reported, thoracic radiographs lacked the sensitivity to detect cardiovascular problems in cattle (32). In 2 of the 17 heifers for which thoracic radiographs were available, cardiomegaly was the only reported anomaly. Radiographic assessment of the cardiac silhouette remained subjective, because there are no published reports of normal heart silhouette size in growing calves. Signs that are classically found in calves with VSD are general heart enlargement, especially the left atrium with hypervascularity of the lung. In the ventrodorsal projection, the right heart border can reach the inner surface of the right chest wall (32). However, thoracic radiographs appeared to have been essential in identifying pneumonia in this case series.

Our study suggests that a few cattle with VSDs may remain asymptomatic for a long time. Three heifers out of 25 were presented for a problem not related to the cardiorespiratory system. Two of them, 5 and 6 mo old, were presented for acute bloating. The third was presented at 21 mo with poor breeding ability but without cardiorespiratory clinical signs, other than a heart murmur. In dogs (33), as in human infants (20,34), spontaneous closure of small defects have been reported. In humans, the rate spontaneous closure of the defect during infancy and adolescence was estimated recently at 23%, but it can vary depending of the study (34). In the 2 cases that were reassessed 8 and 17 mo after the 1st ultrasounographic examination, an increase in the size of the VSD was observed. This enlargement of the defect could be related to growth, but it has not been previously reported. Unfortunately, no objective prognostic indicators have been identified concerning the outcome of cattle with this condition.

Prognosis for most of the affected calves was poor. The retrospective nature of this study could explain this finding. Prognosis could have been worsened if the owner was informed that VSD could be a genetic problem or that calves with a VSD may never become productive animals, thereby influencing his decision to euthanize them after diagnosis. Of the 6 heifers that were discharged from the CHUV and for which follow up information was available, only 1 survived and was pregnant at 24 mo of age. Poor reproductive performance was the most common complaint and reason for early culling in 4 heifers. Growth retardation was associated with this problem in 4 heifers. However, genital tract abnormalities were not investigated. Genital tract malformation associated with VSD seem to be rare, although uterus unicornis has previously been described in a heifer with VSD (8). Necropsies performed in 15 calves revealed no genital tract anomaly in our study.

Ventricular septal defect may be underestimated in general practice. Pulmonary infection can be a confounding factor which can mask the characteristic right sided pansystolic murmur. Cardiac ultrasonography was the procedure of choice to obtain a final diagnosis. Size of the defect did not seem to be of prognostic value in this study. Other ultrasonographic parameters, such as velocity of the blood through the defect or the ratio of the size of the VSD on the size of the aortic root, used in other species, were not investigated. Further studies need to be designed prospectively with standardized ultrasonographic protocols in order to better understand the disease in calves. Analysis of pedigrees and prediction of the outcome could be determined, if the owner desired to keep the affected animal alive for breeding purpose only. CVJ

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